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Ischemic Heart Disease and Spousal Smoking in the National Mortality Followback Survey[1]

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Data on never-smoking decedents from the 1986 National Mortality Followback Survey were used to perform a case–control analysis of ischemic heart disease in relation to spousal cigarette smoking. The case groups consisted of 475 men and 914 women who died from heart disease. Controls consisted of 988 men and 1930 women who died from other causes. In this study there was no association between spousal smoking and ischemic heart disease in either sex (males, odds ratio = 0.97; females, odds ratio = 0.99). The results of this study are in striking disagreement with risk elevations reported in several previous studies of spousal smoking and heart disease. © 1995 Academic Press, Inc.

INTRODUCTION

Several epidemiologic studies have reported associations between ischemic heart disease mortality in never-smokers and exposure to environmental tobacco smoke (ETS) from spousal smoking (Hirayama, 1984; Garland *et al.*, 1985; Lee *et al.*, 1986; Martin *et al.*, 1986; Svendsen *et al.*, 1987; Butler, 1988; Helsing *et al.*, 1988; Palmer *et al.*, 1988; He, 1989; Hole *et al.*, 1989; Jackson, 1989; Humble *et al.*, 1990; Dobson *et al.*, 1991; LaVecchia *et al.*, 1993; He *et al.*, 1994). Despite the high rate of heart disease death among never-smokers, most of those studies have included relatively few cases; only three studies (Hirayama, 1984; Helsing *et al.*, 1988; Dobson *et al.*, 1991) included substantially more than 100 cases. Although some review panels concluded that there was insufficient evidence to infer that ETS exposure increases the risk of heart disease (National Research Council, 1986; U.S. Surgeon General, 1986), other reviewers have drawn such an inference (Wells, 1988; Glantz and Parmley, 1991; Steenland, 1992; Taylor *et al.*, 1992).

The National Mortality Followback Survey (NMFS), which was based on a probability sample of adult deaths in the United States in 1986, offered the opportunity to perform a case–control analysis of ischemic heart disease and spousal smoking that includes more never-smoking cases than any previously published study. This paper reports the results of that case–control study.

MATERIALS AND METHODS

The National Mortality Followback Survey was conducted in 1986 by the U.S. National Center for Health Statistics (NCHS) (NCHS, 1988; Seeman *et al.*, 1989). The survey was based on a national probability sample of about 1% of all deaths in 1986 of U.S. residents aged 25 years or older. This sample was drawn from the Current Mortality Sample (CMS), a 10% sample of death certificates that NCHS receives from state authorities approximately 3 months after the deaths occur. The NMFS sample included data from 49 states and the District of Columbia; deaths from Oregon were not included because of respondent consent requirements.

Of the 18,733 death certificates in the NMFS sample, about 2500 were selected with certainty from the CMS for certain causes of death and population subgroups. These included all deaths due to ischemic heart disease (International Classification of Diseases, Revision 9, codes 410–414) for males aged 25–44 years and for females aged 25–54 years. In addition, some population groups were oversampled. For example, black decedents were oversampled 2.9 times and those under 55 years of age were oversampled 3.1 times.

Next of kin of decedents in the NMFS sample were asked to complete a questionnaire that included questions on demographic characteristics, dietary patterns, cigarette smoking (personal and spousal), alcohol consumption, education, income, and history of

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TABLE 1

Distribution of Male and Female Ischemic Heart Disease Cases and Controls by Race and Mean Ages at Death (National Mortality Followback Survey, 1986)

	Cases		Controls		Test for case–control difference
	No.	%	No.	%	
Males					
Race					
White	356	74.9	681	68.2	
Black	96	20.2	264	26.5	$P = 0.02$
Other	23	4.9	53	5.3	
Mean age at death	72.64		64.76		$P < 0.0001$
Females					
Race					
White	675	73.9	1,339	69.4	
Black	212	23.2	516	26.7	$P = 0.04$
Other	27	2.9	75	3.9	
Mean age at death	78.17		71.89		$P < 0.0001$

diseases. The questionnaire response rate was 88.6%. The NMFS is described in more detail in Seeman *et al.* (1989).

The decedents included in the present study were restricted to those reported by next of kin to be lifetime never-smokers, defined to be those who had never smoked 100 or more cigarettes in their entire lives. Furthermore, decedents were excluded from analysis if they had never married or their marital status was unknown or if it was not known whether their spouse had smoked; 549 male and 692 female subjects were excluded for those reasons. After these exclusions, the case group consisted of 1389 (475 male and 914 female) ischemic heart disease deaths. Deaths from causes generally considered to be smoking related were excluded from the control group. Excluded causes of death were cancers of the lung, mouth, pharynx, larynx, esophagus, pancreas, bladder, kidney, and cervix, cerebrovascular disease, and chronic obstructive lung disease. The control group then consisted of 998 male decedents and 1930 female decedents.

Spousal smoking status was based on answers to two questions asked of questionnaire respondents about the decedents: "Did (any) spouse smoke at least 100 cigarettes?"; and "How many cigarettes a day did (this) spouse smoke?" (If more than one spouse smoked, the answer to the second question was for the most recent spouse.)

Multiple logistic regression methods were used to estimate spousal smoking odds ratios adjusted for age and race (Breslow and Day, 1980). Trend tests were calculated by assigning scores to exposure categories, including the no-exposure category, and adding the quantitative variable so defined to the logistic regression model.

RESULTS

Table 1 presents data on race and age at death for ischemic heart disease cases and controls. For both sexes the race distribution of heart disease cases differed significantly from that of controls, and mean ages at death also differed significantly between cases and controls. All analyses of spousal smoking and heart disease risk were adjusted for age and race.

No overall spousal smoking–heart disease risk elevation was observed for either males or females, the adjusted odds ratios being 0.97 and 0.99, respectively (Table 2), nor was there for either sex any significant trend in risk with increasing numbers of cigarettes smoked per day by the spouse (trend test P values: males, 0.8; females, 0.3).

The spousal smoking results were not appreciably affected by adjustment for history of hypertension, history of diabetes, family history of heart attack, relative weight, alcohol consumption, dietary factors, education, and family income. There were no significant age–spousal smoking interactions.

DISCUSSION AND CONCLUSIONS

In this study there was no suggestion of an association between exposure to ETS from spousal smoking and ischemic heart disease. Odds ratios were below 1.0 for both men (odds ratio = 0.97) and women (odds ratio =

TABLE 2

Odds Ratios for Ischemic Heart Disease Death among Never-Smokers by Spousal Smoking Categories (National Mortality Followback Survey, 1986)

	Cases	Controls	Odds ratio*	95% confidence interval
Males				
Spousal smoking				
No	378	783	1.0	
Yes	97	215	0.97	0.73–1.28
Spouse's cigs/day				
<15	38	107	0.76	0.51–1.14
15–34	45	92	1.07	0.72–1.59
35+	6	12	0.92	0.33–2.55
Trend test			$P = 0.8$	
Females				
Spousal smoking				
No	469	969	1.0	
Yes	455	961	0.99	0.84–1.16
Spouse's cigs/day				
<15	139	336	0.85	0.67–1.07
15–34	224	405	1.15	0.94–1.41
35+	52	111	1.06	0.74–1.52
Trend test			$P = 0.3$	

* Adjusted for age and race.

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0.99), and there was no indication of an increasing trend in risk with increasing numbers of cigarettes smoked by the spouse. Spousal smoking is of course a crude surrogate for ETS exposure, so some misclassification of exposure is present in all studies that use spousal smoking as an exposure index. Also, death certificate data are subject to misclassification of cause of death. If there was an association between ETS exposure and the risk of heart disease, random misclassification of exposure, and of cause of death, would tend to bias an observed association toward the null. However, such biases could not explain the complete absence of association reported here.

The results of the present study are in marked contrast to risk elevations reported in other ETS–heart disease epidemiologic studies (Hirayama, 1984; Garland *et al.*, 1985; Lee *et al.*, 1986; Martin *et al.*, 1986; Svendsen *et al.*, 1987; Butler, 1988; Helsing *et al.*, 1988; Palmer *et al.*, 1988; He, 1989; Hole *et al.*, 1989; Jackson, 1989; Humble *et al.*, 1990; Dobson *et al.*, 1991; LaVecchia *et al.*, 1993; He *et al.*, 1994). Those studies reported sex-specific relative risks ranging from 0.97 to 4.00, with a pooled relative risk of about 1.3 (Glantz and Parmley, 1991). Largely on the basis of published epidemiologic data, Wells (1988), Glantz and Parmley (1991), and Steenland (1992) concluded that exposure to ETS increases the risk of heart disease and estimated that some 50,000 heart disease deaths per year among U.S. nonsmokers are attributable to ETS exposure.

The results of this study are consistent with those of recent analyses of data on spousal smoking and heart disease from two large American Cancer Society cohort studies, Cancer Prevention Study I and Cancer Prevention Study II (CPS-I and CPS-II) (LeVois and Layard, 1995). Neither of those analyses suggested an association between spousal smoking and heart disease; sex-specific relative risks ranged from 0.97 to 1.03. The CPS-I data included 14,898 coronary heart disease (CHD) deaths among never-smokers, and the CPS-II data included 3065 CHD deaths among never-smokers.

The total number of never-smoker heart disease cases in the two CPS studies and the present NMFS study is over 19,000, a far greater number than the total of about 3000 cases in all other published studies of ETS and heart disease. When the six sex-specific relative risks from the CPS studies and the NMFS study are combined, the pooled relative risk is 1.00 (95% confidence interval, 0.97–1.04), which is highly significantly different from the pooled relative risk of 1.3 from previous studies (LeVois and Layard, 1995). The inconsistency between the results of the three new spousal smoking–heart disease analyses and those of previous published studies suggests that the 30% risk elevation estimated from previous studies can be partly or wholly explained by publication bias. Whatever the explanation for the inconsistency, taken as a whole the ETS–heart disease epidemiology clearly does not justify an inference that ETS exposure increases heart disease risk, and estimates of never-smoker heart disease deaths attributable to ETS exposure are without a valid basis.

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